U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 22 February 2006

CASE NO.: 2005-BLA-5115

In the Matter of:

KATHRYN S. BARKER, Widow of
JAMES A. BARKER,
Claimant,

v.

PEABODY COAL COMPANY,
Employer,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest.

Appearances: Sandra M. Fogel, Esquire
For the Claimant

Scott A. White, Esquire
For the Employer

Before: STEPHEN L. PURCELL
Administrative Law Judge

DECISION AND ORDER AWARDING BENEFITS

This proceeding arises from a request for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq*. In accordance with the Act and the pertinent regulations, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs for a formal hearing.

A formal hearing was conducted in Carbondale, Illinois on April 12, 2005 at which time all parties were afforded a full opportunity to present evidence and argument as provided in the Act and the regulations issued thereunder, found at Title 20, Code of Federal Regulations. During the hearing, Director's Exhibits ("DX") 1 through 40, Claimant's Exhibit ("CX") 1 and Employer's Exhibits ("EX") 2, 4-8, and 10-12 were received in evidence while a ruling on EX 1, 3 and 9 was reserved pending the submission of post-hearing briefs by the partiers. Hearing

Transcript ("Tr.") 9-10, 48. EX 1 and 9 were thereafter admitted and EX 3 was excluded pursuant to my orders dated July 21, 2005, August 26, 2005, and September 22, 2005. Since EX 12, the deposition testimony of Dr. Renn, would only be admissible if Dr. Renn's medical report had not been excluded, *see* 20 C.F.R. § 725.414(c), that exhibit is also excluded and will not be considered in this survivor's claim. The remaining evidence described above has been made part of the evidentiary record.

In my order of September 22, 2005, the parties were ordered to file closing briefs on or before October 21, 2005. Both Employer and Claimant thereafter filed written arguments.

ISSUES

Employer and the Director have both conceded that Claimant is an eligible survivor of the Miner, and that the Miner worked at least ten years in underground coal mining. DX 40, Tr. 6, 32-33, 41. Employer further stipulated that it was the Responsible Operator and that the individual upon whom the claim was based was a miner. Tr. 5. The remaining issues presented in this claim are whether the Miner had pneumoconiosis arising out of coal mine employment and whether the Miner's death was due to pneumoconiosis. DX 40, Tr. 5-6.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History and Factual Background

The Miner, James A. Barker, (hereinafter "the Miner" or "Mr. Barker"), filed an application for benefits on January 9, 1980. DX 1. It was denied first by the District Director and then by Administrative Law Judge Lawrence E. Gray in a May 19, 1986 decision and order. *Ibid*. Judge Gray's decision denying benefits was thereafter affirmed by the Benefits Review Board in an October 31, 1989 decision. *Ibid*. A petition for modification was then filed by the Miner on April 29, 1991, the petition was denied by the District Director, and Judge Gray subsequently issued a decision and order on August 4, 1993 in which he found the modification petition to be untimely. *Ibid*. The Board thereafter vacated Judge Gray's decision and remanded the claim for reconsideration. The case was then transferred to Administrative Law Judge Frank D. Marden who, on January 24, 1996, issued a decision and order denying modification based on his finding that the Miner had not established the existence of pneumoconiosis. The Board thereafter affirmed Judge Marden's decision on April 24, 1997. *Ibid*.

The Miner died on January 22, 1999, and on June 7, 2003, Kathryn S. Barker, the Miner's widow, (hereinafter "Claimant" or "Mrs. Barker"), filed an application for survivor's benefits. DX 3, 9. Her application was denied by the District Director on July 8, 2004, and on July 26, 2004, Claimant requested a formal hearing. DX 34, 36. This matter was thereafter referred to the Office of Administrative Law Judges on October 14, 2004. DX 40. A formal hearing was conducted on April 12, 2005 in Carbondale, Illinois.

Mr. Barker was born on June 19, 1937, and he died on January 22, 1999 at the age of 61. DX 3, 9, Tr. 12. Mrs. Barker was married to the Miner on November 30, 1973, they were briefly

divorced after ten years for a period of approximately five months, were remarried on February 1, 1984, and they then remained married until the Miner's death. DX 3, 7, 8, Tr. 33. Mrs. Barker has not remarried since her husband died. Tr. 33.

According to Claimant, Mr. Burns' clothes and lunch bucket were covered with coal dust when he returned from work at the end of his shift during the approximately 11 years that he worked as an underground coal miner. Tr. 33-34. She further testified that he began having problems with breathing and coughing beginning in about 1975. Tr. 34-35. Mrs. Barker and her husband used to enjoy dancing, and her husband regularly worked in the yard mowing and trimming the grass, but he stopped both activities around 1982 because he would become short of breath. Tr. 35-36. His breathing problems got drastically worse after he retired, and he was treated by Drs. Cavenus, Watters, and Walker. Tr. 36-37. His treatment included taking pills, and the use of inhalers and a nebulizer up to four times per day. Tr. 37. He began nebulizer treatments around 1990 and was placed on supplemental oxygen in 1992 or 1993. Tr. 38. He underwent lung reduction surgery in 1995, and, although he was able to stop using the nebulizer, inhaler, and oxygen for a while, he had to go back to using them around 1997. *Ibid*. The Miner was smoking about a pack of cigarettes a day in 1973 when he and Mrs. Burns got married, and he quit smoking in June 1979 when he retired and was diagnosed with lung disease. Tr. 38-39. Mr. Barker experienced complications after his 1995 lung surgery and went into respiratory failure. Tr. 40. He was hospitalized for about a month. *Ibid*. He underwent hip surgery on January 12, 1999 but could not be given general anesthesia because of his lung condition. Tr. 41. He was sent to a nursing home for therapy and rehabilitation following his hip surgery, and he died shortly thereafter. *Ibid*.

Length of Coal Mine Employment

As noted above, Employer has stipulated to at least ten years of coal mine employment. When the Miner previously filed for benefits, the Director found, and Employer stipulated to, 10 years and 10 months of coal mine employment. DX 1. Based upon the documented evidence of record, I find that James A. Barker was a coal miner within the meaning of § 402(d) of the Act and 20 C.F.R. § 725.202 of the regulations for at least 10 years. He last worked as a coal miner in 1979.

Responsible Operator

The evidence reveals, and Employer concedes, that the Miner worked as a coal miner for Peabody Coal Company between 1968 and 1979. DX 1, 4-6. Accordingly, I find that it was properly designated the responsible operator in this case.

Applicable Law

Because this claim was filed after the enactment of the Part 718 regulations, entitlement to benefits will be evaluated under the Part 718 standards. 20 C.F.R. § 718.2. In order to establish entitlement to benefits under Part 718, Claimant must establish by a preponderance of the evidence that the Miner's death was due to pneumoconiosis. *See generally Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994).

Death Due to Pneumoconiosis

Section 718.205 provides that benefits are available to eligible survivors of a miner whose death was due to pneumoconiosis. An eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that the miner's death was due to pneumoconiosis;

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or where death was caused by complications of pneumoconiosis; or

3. Whether the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a substantially contributing cause of a miner's death if it hastens the miner's death. 20 C.F.R. § 718.205(c)(5).

In a survivor's claim, a threshold determination as to the existence of pneumoconiosis must first be made, *See 20 C.F.R. §718.205(a)(1); Trumbo v. Reading Anthracite Co.*, 17 BLR 1-84 (1993). The evidence relevant to the issues presented is set forth below.

Medical Evidence

Neither party offered as "initial evidence" under 20 C.F.R. § 725.414 any chest x-ray, pulmonary function study, or arterial blood gas study evidence in the survivor's claim. The record contains one autopsy report,[1] four medical reports, a death certificate, and assorted treatment and hospitalization records[2] which are described below.

Autopsy Report of Dr. John A. Heidingsfelder

A report dated January 23, 1999 by Dr. Heidingsfelder notes that he conducted "a postmortem anatomic procedure . . . at the request of the deceased's next of kin Kathy Barker." DX 15 at 4. External examination revealed, *inter alia*: evidence of recent right hip surgery; fibrous adhesions of both right and left pleural surfaces; regions of dense fibrous scarring associated with linear fragments of apparent surgical material in both right and left upper

[1] At the formal hearing, Employer offered a June 20, 2005 report by Dr. Oesterling (EX 1) as an "autopsy" report pursuant to 20 C.F.R. § 725.414(a)(3)(i). I subsequently found the report was a medical report rather than an autopsy report for the reasons set forth in my orders dated July 21, 2005 and August 26, 2005. The report was thereafter accepted in evidence as one of Employer's two medical reports permitted under the regulations.

[2] The treatment records include, *inter alia*, a report of an October 10, 1995 biopsy of the Miner's right and left lung noting a diagnosis of emphysema. DX 14 at 13.

portions of the chest and lung region which were about 1 cm wide, and several centimeters long with focal areas of multiple metallic staple formation; associated fibrous scarring in the upper lung and chest regions; an absence of pericardial fibrous adhesions in the pericardial sac (a finding atypical of a cardiac procedure); and a mildly enlarged heart, particularly in the region of the left ventricle. *Id.* at 4-5. With respect to the lungs, Dr. Heidingsfelder noted:

> The lungs reveal a diffuse pattern of dense fibrous adhesions over all pulmonary lobes. There is associated moderate boggy texture to palpation of the lungs, particularly and more prominent in the lower lobes bilaterally. . . . Sections of the lung tissue reveals [sic] anthracotic pigment deposition, focal moderate to marked and focal regions of pulmonary edema and congestion. . . . Opening of the main stem bronchi bilaterally reveals there to be present a moderate pitting and trenching of the bronchial mucosa. No foreign body obstruction of the airways is seen. No frank bullous emphysematous changes are seen. . . .

Id. at 6. He further noted, with respect to his anatomic findings, the presence of moderate to marked focal pulmonary anthracosis, moderate pulmonary edema and congestion bilaterally in the lower lobes of the lung, extensive pleural fibrous scarring and adhesion formations bilaterally in the upper lobes of the lungs, lymph node anthracosis of the peritracheal and perihilar lymph nodes, and atherosclerotic cardiovascular disease. *Id.* at 7. In a section of his report captioned "comment," Dr. Heidingsfelder wrote, *inter alia*, that Mr. Burns had moderate to marked pulmonary anthracosis, significant changes within the bronchi consistent with chronic bronchitis, and evidence of both old and recent heart attacks. He concluded based on his anatomic examination:

> The patient may well have died of a probably acute myocardial infarct due to atherosclerotic cardiovascular disease. Other significant conditions which may have contributed to the timing of death would include the patient's coworkers [sic] pneumoconiosis. Microscopic examination is pending at this time.

Id. at 9. Dr. Heidingsfelder's findings after microscopic examination of tissue slides included pulmonary emphysema, pulmonary anthracosis with focal interstitial fibrosis, lymph nodal anthracosis, moderate to marked pulmonary edema and congestion with intra-alveolar recent hemorrhage, and evidence of an acute myocardial infarct. *Id.* at 13.

Claimant's counsel deposed Dr. Heidingsfelder regarding his opinions in this matter on June 23, 2000. DX 15 at 14-71. He testified that, based on his macroscopic and microscopic findings at autopsy, Mr. Barker suffered from coal workers' pneumoconiosis caused by coal mine employment. *Id.* at 30. He further testified:

> He has significant degrees of chronic bronchitis; he has significant degrees of pulmonary emphysema, which diseases can be associated with coal workers' pneumoconiosis. There are lesions of localized emphysema which are a more characteristic feature relating to coal workers' pneumoconiosis. It's hard for me to separate out that part which is specifically related to his mining exposure versus other types of exposure that he may have had in his lifetime.

Id. at 31. Dr. Heidingsfelder could not determine the cause of Mr. Barker's emphysema or chronic bronchitis, both of which are forms of chronic obstructive pulmonary disease ("COPD"), stating that it was "[p]erhaps environmental exposure, perhaps smoking." *Id*. at 31-32. He opined that the overall severity of the Miner's pulmonary disease was "[a]t least moderate severity" and further stated that he was not sure he could separate Mr. Barker's pneumoconiosis from his emphysema and chronic bronchitis. *Id.* at 33. Dr. Heidingsfelder stated that the Miner's chronic lung disease "may well have aggravated or contributed to or hastened the timing of his death." *Id.* at 35. He subsequently testified that he believed, within a reasonable degree of medical certainty, that pneumoconiosis, emphysema, and bronchitis, were causative factors in, and hastened, Mr. Barker's death. *Id.* at 35-36. Dr. Heidingsfelder also testified that because of the duration of Mr. Barker's heart attack, occurring over a period of a few days to a few weeks, the Miner's pulmonary impairment "in all reasonable probability would have contributed to the time of his death due to his heart attack." *Id.* at 39.

On cross-examination, Dr. Heidingsfelder acknowledged that he had no independent knowledge of Mr. Barker's medical, social or work history, and that his opinions were based solely on the autopsy he performed. *Id.* at 42. With respect to his findings regarding coal workers' pneumoconiosis, Dr. Heidingsfelder testified that those findings were based on "the presence of other chronic lung diseases of emphysema and bronchitis, the presence of diffuse pleural fibrous scarring, the presence of focal interstitial fibrosis within lung tissues, the presence of marked degrees of the carbon pigment deposits within lung and lymph nodal tissues, and the presence of lesions of localized emphysema characteristic of simple coal workers' pneumoconiosis." *Id.* at 44-45. He further testified that the "localized emphysema around an anthracotic macule" he observed in the Miner's lungs was seen only in simple coal workers' pneumoconiosis, but the other findings were not specific solely to that disease. *Id.* at 45, 48.

On re-direct examination, Dr. Heidingsfelder clarified that, when he said he could not determine the relative contribution of emphysema and bronchitis, versus pneumoconiosis, to Mr. Barker's myocardial infarction, it was because the effects of emphysema, bronchitis, and pneumoconiosis could not be separated from each other. *Id.* at 65-66. He further testified that, based on his examination of the Miner's heart and lungs, it was his opinion that Mr. Barker died of a heart attack which occurred over a period of days or weeks and that chronic lung disease, which included emphysema, bronchitis, and pneumoconiosis all working together, was a contributing factor to the timing of his heart attack. *Id.* at 67-68.

Medical Certificate of Death

Dr. Roger Watters signed the death certificate dated January 22, 1999 noting "acute myocardial infarction" as the immediate cause of death and listing "pneumoconiosis" as a significant condition contributing to death. DX 9.

Physician Opinion of Dr. Everett F. Oesterling

Dr. Oesterling reviewed available medical evidence, including biopsy and autopsy slides, submitted to him by Employer's counsel and thereafter prepared a written report of his findings

and conclusions dated June 25, 2004. EX 1. He found microscopic evidence of mild to moderate macular coal workers' pneumoconiosis. *Id.* at 2. He concluded that the Miner's pulmonary impairment "cannot be attributed to this minimal structural change" and that pneumoconiosis was not a substantial or contributing cause of Mr. Barker's death and that his death was not hastened in any way by pneumoconiosis. *Ibid*. Dr. Oesterling concluded that Mr. Barker's pulmonary disability was due to "severe panlobular leading to bullous pulmonary emphysema involving both of [the Miner's] upper lobes." *Id.* at 3. He found "no pathologic evidence of an association between exposure to coalmine [sic] dust and the evolution of this significant respiratory disease." *Ibid*. According to Dr. Oesterling:

> The role of cigarettes in the evolution of emphysema is well established within the literature, most recently in an executive summary posted on the Surgeon General's web page concerning the effects of smoking in the American population. . . .Obviously the smoking history [of Mr. Barker] set forth within the medical records including the comments concerning asthma fairly explain the reason that this gentleman developed his severe pulmonary emphysema.

Id. at 4. Examination of lung tissue revealed evidence of pulmonary hemosiderosis and marked passive pulmonary congestion, neither of which, according to Dr. Oesterling, were attributable to coal dust exposure. *Id.* at 5-6. Dr. Oesterling concluded that Mr. Barker's "most significant disease process resulting in his death can be attributed solely to his heart disease and resultant impact on this gentleman's remaining lung tissue." *Id.* at 7. He further stated that: the Miner's death was a result of cardiopulmonary arrest due to arteriosclerotic coronary vascular disease; Mr. Burns suffered from repeated bouts of severe passive pulmonary congestion prior to death caused by ischemic changes which resulted in interstitital pulmonary fibrosis; and none of these disease processes were attributable to coal mine dust exposure. *Ibid*.

Physician Opinion of Dr. Peter G. Tuteur

Dr. Tuteur reviewed available medical evidence, including biopsy and autopsy slides, submitted to him by Employer's counsel and issued a report dated February 7, 2005 in which he reported his findings and conclusions. EX 9. He noted that the Miner worked in underground coal mining for eleven years and "was exposed to sufficient amounts of coal mine dust to produce coal workers' pneumoconiosis in a susceptible host." *Id.* at 3. He further noted that he smoked cigarettes for twenty years ending in 1979 at a rate reflected in various medical records of between one and as many as five packs of cigarettes per day. *Ibid*. According to Dr. Tuteur, the Miner experienced several major health problems during his life including coronary artery disease, myocardial infarctions, emphysema, acute bronchitis, pneumonia, hemoptysis, and degenerative joint disease. *Id.* at 3-4. He concluded, based on pulmonary function studies, that Mr. Barker had a moderate obstructive ventilatory defect in 1979 which worsened into a severe obstructive defect by 1991 through early fall of 1995. *Id.* at 4. Dr. Tuteur further concluded that lung reduction surgery resulted in "a dramatic and immediate improvement of FEV-1 . . . which remained stable then for the next three years." *Ibid*. According to Dr. Tuteur, it was only following his hip replacement surgery that Mr. Barker again experienced a worsening of his obstructive ventilatory defect. *Ibid*. Dr. Tuteur further wrote:

> At the time of lung volume reduction surgery, resected lung tissue was evaluated microscopically and found predominantly if not exclusively to be emphysema. Following the death in 1999, an autopsy was performed by Dr. Heidingsfelder who felt that not only was emphysema, pulmonary edema, and congestion present, but also the diagnosis of simple coal workers' pneumoconiosis could be made at the pathologic level. Yet, neither the report of the autopsy, nor the transcript of Dr. Heidingsfelder's deposition convincingly demonstrated that the standard pathologic criteria for the diagnosis of coal workers' pneumoconiosis were in fact met. Almost certainly the cause of death was an acute myocardial infarction.
>
> Based on this information, there is no convincing data to indicate the presence of neither [sic] medical nor legal coal workers' pneumoconiosis of sufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary function, radiographic change or fulfillment of pathologic criteria. Mr. Barker likely died suddenly in the perioperative period following hip replacement surgery with a myocardial infarction. Complicating his death almost certainly was the advanced chronic pulmonary emphysema despite functional improvement after lung volume reduction surgery. Neither the coronary artery disease, nor the chronic obstructive pulmonary disease, was related to the inhalation of coal mine dust or the development of coal workers' pneumoconiosis. . . .

Id. at 4-5. With respect to coal workers' pneumoconiosis, Dr. Tuteur noted that he would expect to find evidence of reduced total lung capacity reflecting a restrictive abnormality and persistent irreversible impairment of gas exchange for the disease to produce impairment of pulmonary function. *Id.* at 6-7. He found no such evidence in the medical records of Mr. Barker. *Id.* at 7. Dr. Tuteur further explained:

> It should be recognized that the inhalation of coal mine dust may produce airflow obstruction even in the absence of simple coal workers' pneumoconiosis and in the absence of a positive x-ray. . . . [T]he COPD phenotype occurs infrequently among never smoking miners (almost certainly less than 1% of the time) but relatively quite frequently (perhaps 20% of the time) among cigarette smoking never miners. Thus, with reasonable medical certainty, in the case of Mr. James A. Barker his COPD phenotype is cigarette smoke-induced, not coal mine dust related.

Ibid. Dr. Tuteur concluded based on all the medical evidence that the Miner "did not have coal workers' pneumoconiosis or any other coal mine dust-related disease process of sufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary function, or radiographic change." *Id.* at 12. He further concluded that the Miner had "severe and advancing chronic obstructive pulmonary disease, predominantly emphysema . . . [which was] directly related to and caused by the inhalation of tobacco smoke and [was] unrelated to, not aggravated by, and not caused by the inhalation of coal mine dust." *Ibid*.

Dr. Tuteur was deposed by Employer's counsel on April 5, 2005 regarding his opinions in this case. EX 11. He noted that subsequent to his report, he reviewed the March 11, 2005 report of Dr. Green. *Id.* at 15. It was then his conclusion that "simple coal workers' pneumoconiosis was present at the pathologic level" but he testified that "additional data is required to attempt to identify the degree of severity and profusion." *Id.* at 16. He believed the Miner's eleven years in underground coal mining resulted in "sufficient exposure in a susceptible host to produce coal workers' pneumoconiosis and other coal mine dust-related disease processes, such as what I will call COPD phenotype." *Id.* at 18. He further testified:

> [M]edical records indicate that Mr. Barker smoked up to five packages per day for in excess of 20 years. So we are comparing up to 150 pack years versus 11 years of working in the coal mine industry.
> . . . . .
>
> He did have chronic obstructive pulmonary disease, which I recognize can be caused by the inhalation of coal mine dust, but in this case, in this very heavy-smoking gentleman, was due to the chronic inhalation of tobacco smoke, not coal mine dust. . . . .

Id. at 18, 21. With respect to his opinion that inhalation of coal mine dust may produce a "COPD phenotype" in some people, Dr. Tuteur testified that various medical studies have led him to conclude that the frequency with which this occurred was "substantially less than one percent." *Id.* at 26-27. In contrast, according to Dr. Tuteur, the frequency with which smokers developed COPD was about 20 percent. *Id.* at 27. He thus "concluded that, with reasonable medical certainty, Mr. Barker's COPD phenotype was due to the chronic inhalation of tobacco smoke, up to 150 pack years, for – rather than coal mine dust." *Ibid*. Dr. Tuteur acknowledged that he disagreed with the U.S. Department of Labor and NIOSH regarding the prevailing view in the medical community with respect to his conclusions. *Id.* at 41.

Physician Opinion of Dr. Francis H.Y. Green

Dr. Green reviewed the available medical records and autopsy slides of Mr. Barker submitted to him by Claimant's counsel and thereafter prepared a written report of his findings and conclusions dated March 11, 2005. CX 1. He found that the slides he reviewed revealed "widespread evidence of coal workers' pneumoconiosis, comprising macules, micronodules and interstitial fibrosis with pigmentation." *Id.* at 2. He further concluded that "the number and profusion of the lesions would be consistent with a severe form of simple coal workers' pneumoconiosis." *Ibid*. In addition to pneumoconiosis, Dr. Green found "widespread severe panacinar emphysema, as well as chronic bronchitis involving the large airways and chronic bronchiolitis involving the smaller airways. *Ibid*. His review of sections of the heart revealed evidence of "old myocardial infarctions and a large acute myocardial infarction involving the left ventricular wall." *Ibid*. Dr. Green agreed with the physician completing Mr. Barker's death certificate (Dr. Watters) and the pathologist who conducted the autopsy (Dr. Heidingsfelder) that "chronic lung disease was a contributing factor to the [Miner's] death." *Id.* at 3. He concluded that the Miner's clinical pneumoconiosis was due entirely and exclusively to his exposure to coal

mine dust. *Id.* at 4. With respect to the cause of Mr. Barker's COPD (emphysema and bronchitis) he wrote:

> Chronic bronchitis and pulmonary emphysema, together comprising the clinical syndrome of [COPD], can be caused by a number of factors, the most common of which are exposure to cigarette smoke and exposure to occupational dusts and fumes. Mr. Barker developed his pulmonary impairment at a relatively young age, and there was a family history of emphysema, raising the possibility that he may have had a genetic predisposition to the development of emphysema. As far as I can tell, the relevant tests were not conducted. In addition to coal mine dust exposure, Mr. Barker also had a history of cigarette smoking, consuming approximately one pack a day for 20 years until he quit in 1979. Numerous epidemiologic and pathologic studies have now unequivocally linked coal mine dust exposure to the development of pulmonary emphysema . . . . Some of these studies have calculated the proportion of emphysema (or reductions in FEV1) attributable to coal dust and cigarette smoking. These show, in general, that one pack year of smoking contributes approximately the same amount to the emphysema score as does one year of underground mining. Using this rule of thumb, it would appear that smoking contributed slightly more to the development of his emphysema than did his exposure to coal mine dust. However, it should be borne in mind that he quit smoking in 1979 and thus for the last 20 years of his life smoking would not further contribute to the development of the emphysema. This would not be true for the relationship with coal mine dust exposure as the dust is retained in the lungs for the lifetime of the individual and continues to irritate the lungs, leading to progression of the emphysema. Taking this into account, I would estimate that smoking and coal mine dust exposure contributed approximately equally to Mr. Barker's emphysema.

Id. at 4. Dr. Green concluded that Mr. Barker had both medical pneumoconiosis (macules, macronodules and interstitial fibrosis) and legal pneumoconiosis (COPD attributable to coal dust exposure) and that this pneumoconiosis "contributed to his death and/or hastened his death by significantly reducing the oxygen supply to the ischemic heart muscle." *Id.* at 4-5.

Hospital and Treatment Records

(1) *1997-1998 Treatment Records of Dr. Roger D. Watters (DX 11)*

Dr. Watters' treatment records contain various objective test results including chest x-ray reports, pulmonary function and blood gas studies, and electrocardiogram results for 1997 and 1998. DX 11. These records and various treatment notes reflect diagnoses of, *inter alia*, diffuse bullous pulmonary emphysema of the upper lobes, interstitial pulmonary opacities compatible with occupational lung disease, chronic obstructive pulmonary disease due to emphysema, hilar and parenchymal calcified granulomatous nodules, arteriosclerotic heart disease, hypertension, and osteoarthritis of the back and hips. *Ibid*.

(2) *1991-1995 Treatment Records of Dr. Edward N. Moore (DX 12)*

Medical records obtained from Dr. Moore reflect, *inter alia*, the following:

8-22-91 examination report noting the Miner is status post anteroapical myocardial infarction and has "severe restrictive and obstructive lung disease." DX 12 at 23.

12-5-91 examination report noting diminished breath sounds with diffuse crackles, no definite wheezes, and impressions of, *inter alia*, "advanced COPD of mixed type . . . [c]oal dust exposure has to be an additional aggravating factor." DX 12 at 24.

8-9-91 hospital admission report noting impressions of acute anterior wall infarction within the past 12 to 24 hours, history of severe COPD and emphysema, and hypertension. DX 12 at 19; *see also* EX 6 at 104.

8-12-91 echocardiographic report noting, *inter alia*, the Miner's evidence of an apical myocardial infarction and normal left and right ventricular ejection fractions. DX 12 at 8.

1-20-94 office visit noting impressions of coronary artery disease and stable from cardiac standpoint. DX 12 at 17.

12-12-94 letter to Dr. Watters from Dr. Alec Patterson stating, *inter alia*, the Miner suffers from advanced chronic obstructive lung disease due to smoking and is a suitable candidate for lung volume reduction. DX 12 at 12-13.

1-16-95 office visit noting the Miner "was considering undergoing partial pneumonectomy for black lung but elected not to at this point." DX 12 at 15.

5-18-95 office note stating the Miner has decided to have lung volume reduction surgery. DX 12 at 11.

5-22-95 report of dobutamine stress echocardiogram noting final impression of previous completed infarction at the apex and evidence of basal inferior wall hypokinesis at rest with high dose Dobutamine induced ischemia. DX 12 at 7.

(3) *1999 Medical Records of Marion Memorial Hospital (DX 13)*

The records of Marion Memorial Hospital include various chest and hip x-ray reports, a pulmonary function study, and a bilateral renal ultrasound report. DX 13.

(4) *1991-1998 Medical Records of Washington University Medical Center and Harrisburg Medical Center (DX 14)*

Records obtained from Washington University Medical Center and Harrisburg Medical

Center include various chest x-ray, ultrasound, pulmonary function study, and other test results as well as, *inter alia*:

> 8-9-91 discharge summary from Harrisburg Medical Center noting diagnoses of inferior wall MI, ASHD, hypertensive crisis, and COPD. DX 14 at 139.
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> 12-3-92 discharge summary from Harrisburg Medical Center noting diagnoses of acute bronchitis, exacerbation of COPD, sinus tachycardia, and hypertension. DX 14 at 136; *see also* EX 6 at 88..
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> 2-17-93 discharge summary from Doctors Hospital of Harrisburg, Inc. noting diagnosis of acute influenza and bronchitis. DX 14 at 126; *see also* EX 6 at 125.
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> 4-16-93 discharge summary from Harrisburg Medical Center noting diagnoses of acute bronchitis, exacerbation of COPD, arteriorsclerotic heart disease, and hypertension. DX 14 at 116.
>
> 3-29-94 discharge summary from Harrisburg Medical Center noting diagnoses of acute bronchitis and exacerbation of COPD. DX 14 at 109; *see also* EX 6 at 59..
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> Report of pre-rehabilitation program for lung reduction surgery beginning 7-31-95 and ending 9-29-95. DX 14 at 107-08.
>
> 10-10-95 biopsy report of right and left lung noting diagnosis of emphysema. DX 14 at 13.
>
> 10-28-95 hospital discharge summary noting bilateral volume reduction and cardiac catheterization were performed. DX 14 at 72. Principal and secondary diagnoses were chronic obstructive pulmonary disease and coronary artery disease. *Ibid*.
>
> 11-18-95 letter from Dr. Cooper noting the Miner underwent median sternotomy and bilateral lung volume reduction surgery on October 10, 1995 to relieve "some of the more distressing symptoms associated with his very severe obstructive lung disease." DX 14 at 5.
>
> 11-20-95 letter from Dr. Mitchell Horowitz noting Miner discharged the preceding week following lung volume reduction surgery. DX 14 at 6. Examination revealed "dramatic improvement" in FEV1 following surgery. *Id.* at 7.
>
> 4-16-96 letter from Dr. Roger D. Yusen noting the Miner was seen six-months after his lung volume reduction surgery. DX 14 at 3. Impression at that time was stable COPD by history and markedly improved after surgery. *Id.* at 4. Continued bronchodilator therapy was recommended, although the result in terms of improved spirometry, oxygenation, and functional status were described as

"outstanding." *Ibid*.

10-30-98 discharge summary from Harrisburg Medical Center noting diagnoses of hemoptysis, COPD, renal insufficiency, anemia, and arteriosclerotic heart disease. DX 14 16 143; *see also* EX 6 at 14..

10-29-98 report of bronchoscopy noting diagnosis of blood coming from right lower lobe of lung, probably secondary to COPD, and no endobronchial lesions seen. DX 14 at 147.

(5) *Medical Records of Drs. Edward Moore, R.E. Hyatt, and Robert Watters (DX 15)*

Medical records obtained by Claimant and submitted to the District Director include various chest x-ray, ultrasound, pulmonary function study, and other test results as well as, *inter alia*:

8-11-91 report of examination by Dr. Thomas K. Browne noting impression of ASHD, history of hypertension and advanced COPD of mixed type. CX 15 at 106. According to Dr. Browne, with respect to the Miner's COPD, "certainly he has bullous emphysema and superimposed some increased airway reactivity by history. This in turn, may be at least partially on an atopic basis. Coal dust exposure has to be an additional aggravating factor." *Ibid*.

8-30-91 letter from Dr. Moore stating Miner has "severe underlying C.O.P.D. complicated by Black Lung Disease." DX 15 at 102.

10-7-92 consultation report by Dr. Hyatt noting major complaints of hoarseness associated with shortness of breath. DX 15 at 117; *see also* EX 4.

10-28-92 follow-up consultation note by Dr. Hyatt noting "extremely severe airway obstruction at a relatively young age." DX 15 at 113; *see also* EX 4.

1-20-94 office visiting with Dr. Moore noting diagnosis of coronary artery disease and Miner "stable from a cardiac standpoint." DX 15 at 99.

1-16-95 office visit with Dr. Moore noting diagnosis of coronary artery disease and Miner "stable from a cardiac standpoint." DX 15 at 95.

5-19-95 office visit with Dr. Moore noting diagnosis of coronary artery disease and Miner "stable from a cardiac standpoint." DX 15 at 93

1997-1997 treatment notes by Dr. Watters reflecting diagnoses of, *inter alia*, COPD, arteriosclerotic heart disease with previous MI, hypertension, allergic rhinitis, hemoptysis, and pneumonia resolved. DX 15 at 198-213

1-12-99 surgical and consultation reports regarding Miner's right total hip

arthroplasty noting chest tightness and shortness of breath following surgery suggestive of angina. DX 15 at 226-32.

(6) *1991-1998 Medical Records of Harrisburg Medical Center (EX 6)*

Medical records from Harrisburg Medical Center obtained by Employer's counsel, many of which are duplicative of those described above, include various chest x-ray, ultrasound, pulmonary function study, and other test results as well as, *inter alia*:

4-23-93 discharge summary noting diagnoses of acute bronchitis, exacerbation of COPD, ASHD, and hypertension. EX 6 at 77.

4-28-93 discharge summary noting diagnoses of adverse side effect from Zoloft, anxiety, depression, COPD, and ASHD. EX 6 at 71.

12-11-97 discharge summary report noting diagnoses of pneumonia, septicemia due to streptococcus viridans, exacerbation of COPD, arteriosclerotic heart disease, and dehydration. EX 6 at 44.

(7) *October 1995 Medical Records of Barnes-Jewish Hospital (EX 7)*

Medical records from Barnes-Jewish Hospital obtained by Employer's counsel include various chest x-ray, pulmonary function study, and other test results relating to the Miner's bilateral volume reduction and cardiac catheterization in October 1995.

(8) *Medical Records of Deaconess Hospital (EX 8)*

Medical records from Deaconess Hospital obtained by Employer's counsel include various chest x-ray, pulmonary function study, and other test results relating to the Miner's treatment there in August 1991 for an acute myocardial infarction and COPD.

DISCUSSION

A. Pneumoconiosis.

As noted above, an initial determination as to the existence of pneumoconiosis must be made. While employer concedes that the evidence establishes the presence of clinical pneumoconiosis, it disputes the presence of legal pneumoconiosis. Employer's Closing Argument ("Emp. Br.") at 25.

The regulations provide four methods for finding the existence of pneumoconiosis: chest x-rays, autopsy or biopsy evidence, the presumptions in §§ 718.304, 718.305 and 728.306, and medical opinions. § 718.202(a)(1)-(4). There is no evidence of complicated pneumoconiosis, and the claim was filed after 1982. Therefore, the presumptions in §§ 718.304, 718.305, and 718.306 are not applicable herein.

The first method provided in the regulations to establish the existence of pneumoconiosis is by chest x-ray evidence. 20 C.F.R. § 718.202(a)(1). There are no chest x-ray interpretations which conform to the requirements of 20 C.F.R. §§ 718.102 and Appendix A to Part 718. Therefore, pneumoconiosis has not been established pursuant to 20 C.F.R. § 718.202(a)(1).

With respect to establishing pneumoconiosis through biopsy evidence, the only biopsy evidence included in the record is the report of an October 10, 1995 biopsy of the right and left lung prior to lung volume reduction surgery which noted a diagnosis of emphysema. DX 14 at 13. The definition of "legal pneumoconiosis" includes any chronic lung disease or impairment so long as it arose out of coal mine employment. 20 C.F.R. § 718.201(a)(2). This evidence thus *may* support a finding of legal pneumoconiosis if the Miner's emphysema is attributable to his coal mine employment. 20 C.F.R. § 718.202(a)(2).

With respect to establishing pneumoconiosis through autopsy evidence, Dr. Heidingsfelder noted anatomic findings of, *inter alia*, moderate to marked focal pulmonary anthracosis and lymph node anthracosis of the peritracheal and perihilar lymph nodes. DX 15 at 7. His microscopic findings similarly included pulmonary anthracosis with focal interstitial fibrosis and lymph nodal anthracosis. *Id.* at 13. Dr. Heidingsfelder was the only autopsy prosector, and his examination report supports a finding of pneumoconiosis.

With respect to establishing the existence of pneumoconiosis by reasoned medical opinion, both Claimant's and Employer's medical experts agree that the Miner suffered from clinical pneumoconiosis. Dr. Green, for example, found "widespread evidence of coal workers' pneumoconiosis, comprising macules, micronodules and interstitial fibrosis with pigmentation." CX 1 at 2. Dr. Oesterling found microscopic evidence of mild to moderate macular coal workers' pneumoconiosis. EX 1 at 2. Dr. Tuteur, after initially concluding that pneumoconiosis was not shown, testified during his deposition that "simple coal workers' pneumoconiosis was present at the pathologic level . . . ." EX 11 at 15.

As noted above, Employer concedes that clinical pneumoconiosis is established by the medical evidence. Emp. Br. at 25. I thus find, based on the medical evidence set forth above, that Claimant has established the Miner suffered from clinical pneumoconiosis prior to his death. For the reasons set forth in the following discussion on causation, I further find that the Miner's COPD was attributable in substantial part to his more than ten years of exposure to coal mine dust and that his COPD thus constitutes legal pneumoconiosis.

B. Pneumoconiosis Causation.

Both the evidence and the presumption at 20 C.F.R. § 718.203(b) support a finding that the Miner's clinical pneumoconiosis arose out Mr. Barker's coal mine employment. Employer does not contest that the Miner's clinical pneumoconiosis arose out of his more than ten years of coal mine employment. However, Employer does contest that Mr. Barker's COPD was caused

by such employment.[3] Emp. Br. at 25. The four medical opinions relevant to this issue are each discussed below.

Dr. Oesterling, who is board-certified in anatomical pathology, clinical pathology, and nuclear medicine, and is a clinical professor of nuclear medicine technology at Allegheny Community College in Allegheny County, Pennsylvania, rendered an opinion on behalf of Employer which includes, *inter alia*, a finding that Mr. Barker had "mild to moderate macular coalworkers' pneumoconiosis." EX 1 at 2; EX 2. He further opined that the Miner had "severe panlobular leading to bullous pulmonary emphysema involving both of this gentleman's upper lobes [which was the disease process responsible for his pulmonary disability]." EX 1 at 3. He found no evidence of any association between exposure to coal dust and the evolution of the Miner's emphysema. *Ibid*. According to Dr. Oesterling, Mr. Barker had "a significant smoking history with a relatively limited period of coal dust exposure." *Ibid*. He further noted that the medical literature has established the significant role of cigarettes in the evolution of emphysema, and concluded that "the smoking history set forth within the [Miner's] medical records including the comments concerning asthma fairly explain the reason that this gentleman developed his severe pulmonary emphysema." *Ibid*. Dr. Oesterling also concluded that the interstitial fibrosis referred to in Dr. Heidingsfelder's autopsy report "would appear to be more related to [pulmonary hemosiderosis] than to coalmine dust exposure." *Id.* at 5.

Dr. Tuteur, who is board-certified in internal medicine and pulmonary disease, and is an associate professor of medicine at Washington University School of Medicine in St. Louis, Missouri, originally rendered an opinion on behalf of Employer which included, *inter alia*, a finding that neither medical nor legal pneumoconiosis was present in the Miner's lungs. EX 9 at 4; EX 10. When he was subsequently deposed in this case, he changed his opinion stating that "simple coal workers' pneumoconiosis was present at the pathologic level." EX 11 at 16. He further testified, however, that "additional data is required to attempt to identify the degree of severity and profusion." *Ibid*. With respect to emphysema, Dr. Tuteur found that it was a complicating factor in Mr. Barker's death, but he further determined that "[n]either the [Miner's]

[3] Prior to the formal hearing, Employer's counsel sought summary judgment in this case based on the doctrine of collateral estoppel. Counsel thereafter sought, and was granted, permission to seek reconsideration of my determination that Employer could not rely on the doctrine of collateral estoppel to preclude Mrs. Barker from establishing that her husband's COPD was caused or aggravated by the inhalation of coal dust. Relying principally on *Donovan v. Thames*, 105 F.3d 291 (6th Cir. 1997), Employer's counsel asserts in his post-hearing brief that collateral estoppel may be applied against Claimant despite the fact that she was not a party to the Miner's claim when it was denied. Emp. Br. at 8. *Donovan* does not help Employer. That case involved a federal civil rights action brought by Terry Donovan against two police officers after he was convicted in state court of resisting arrest, possession of marijuana, and possession of drug paraphernalia. *Id.* at 293. While the court expressly recognized in dicta that Kentucky has approved the use of non-mutual collateral estoppel against a party to a prior suit even though the parties in the subsequent case are not identical, *id*. at 295, Kentucky's recognition of the doctrine had absolutely nothing to do with the court's decision there. In reversing the District Court's grant of a motion for summary judgment filed by the defendant police officers, the Sixth Circuit Court of Appeals simply concluded that the issue of whether the defendants used excessive force in effecting Donovan's arrest was not a necessary component of, and thus was not actually litigated in, Donovan's state criminal trial. *Id*. at 295. Indeed, under other circumstances (i.e. if Donvoan was attempting to prevail in the federal civil rights action on an issue which was actually decided against him in the state case), its use against Donovan would have been proper. Mrs. Barker, however, unlike Donovan, was *not* a party to the prior proceeding, nor, contrary to Employer's conclusory assertion, was she in privity with the Miner with respect to his previously denied claim. For all the reasons set forth in my order denying summary judgment, Employer's request that collateral estoppel be applied in this case remains denied.

coronary artery disease, nor the chronic obstructive pulmonary disease, was related to the inhalation of coal mine dust or the development of coal workers' pneumoconiosis." EX 9 at 5. According to Dr. Tuteur, "with reasonable medical certainty, in the case of Mr. James A. Barker his COPD phenotype is cigarette smoke-induced, not coal mine dust related." *Id.* at 7. He further described the Miner's pulmonary process as

> severe and advancing chronic obstructive pulmonary disease, predominantly emphysema, treated medically and surgically (lung volume reduction surgery). This condition is directly related to and caused by the inhalation of tobacco smoke and is unrelated to, not aggravated by, and not caused by the inhalation of coal mine dust. . . .

Id. at 12. With respect to his conclusion regarding the etiology of Mr. Barker's COPD, Dr. Tuteur relied on a smoking history of this Miner of from one to five packs of cigarettes per day and on various medical studies which found that smokers were at a substantially greater risk of developing COPD than coal miners. EX 11 at 18, 21, 26-27.

While both Dr. Oesterling and Dr. Tuteur recognize that Mr. Barker had severe COPD during his lifetime, neither physician attributes his COPD to the inhalation of coal dust during the more than ten years he worked underground as a coal miner. Instead, both physicians attribute the Miner's COPD solely to cigarette smoking based, in large part, on medical studies that link smoking to COPD. Both physicians ignore, or minimize the importance of, medical studies which also link COPD to exposure to coal dust. Furthermore, Dr. Tuteur bases his opinion on an inflated smoking history for the Miner, while Dr. Oesterling minimizes the impact of Mr. Barker's more than 10 years of underground coal mining, characterizing it as "a relatively limited period of coal dust exposure." EX 1 at 3. For the reasons set forth below, I find that the opinions of Drs. Oesterling and Tuteur are entitled to less weight than the contrary opinions of Drs. Heidingsfelder and Green.

In formulating his opinion, Dr. Oesterling does not discuss at all the substantial medical literature cited by the Department of Labor in connection with its revisions to the Black Lung regulations which concludes that exposure to coal mine dust may give rise to COPD at a rate nearly equal to that of smoking. For example, in its comments regarding the adoption of the revised regulations, the Department notes, in relevant part:

> Drs. Fino and Bahl find no scientific support that clinically significant emphysema exists in coal miners without progressive massive fibrosis, . . . but the available pathologic evidence is to the contrary. Cockcroft evaluated 39 coal workers and 48 non-coal worker controls dying of cardiac causes in 1979. . . . Centrilobular emphysema (the predominant type observed) was significantly more common among the coal workers. The severity of the emphysema was related to the amount of dust in the lungs. These findings held even after controlling for age and smoking habits.

Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969, as Amended,

65 Fed. Reg. 79920, 79941 (Dec. 20, 2000) ("Final Rule") (internal citations omitted).[4] The Department went on to write:

> Similarly, Leigh and colleagues analyzed 886 miners who died between 1949 and 1982. . . . . They found that miners with more years of face work had worse emphysema pathologically. In a subsequent study of 264 underground coal miners exposed to mixed coal and silica dust, Leigh . . . made the following important findings: (1) The extent of emphysema was strongly related to the total coal content of the lung, age and smoking; (2) in miners who were lifelong non-smokers, the extent of emphysema was strongly related to coal content and age; (3) the extent of emphysema was unrelated to lung silica content; and (4) the extent of lung fibrosis was related to silica content. The authors concluded that "these results provide strong evidence that emphysema in coalworkers is causally related to lung coal content."
>
> Ruckley and colleagues achieved similar results in examining the lungs of 450 coal workers to determine the association between coal mine dust exposure and dust-related fibrosis and emphysema. . . . . The authors found emphysematous changes in 72% of miners who smoked, 65% of ex-smokers, and 42% of nonsmoking miners; emphysema scores were higher in patients with increasing evidence of pneumoconiotic disease; and increasing coal lung dust was associated with the presence of emphysema. Forty-seven percent of miners with no fibrotic lesions had emphysema. Ruckley concluded that "the results support the conclusion that the relationship observed between respirable dust and emphysema in coal workers is, in some way, causal."

Id. at 79941-42 (internal citations omitted). The Department also quotes several studies which establish a link between emphysema and bronchitis to coal dust exposure including the following:

> Most evidence to date indicates that exposure to coal mine dust can cause chronic airflow limitation in life and emphysema at autopsy, and this may occur independently of CWP * * * The relationships between hypersecretion of mucus (chronic bronchitis) and chronic airflow limitation (emphysema) on the one hand and environmental factor of coal mining exposure on the other appear to be similar to those found for cigarette smoking.

Id. at 79939. Dr. Oesterling's failure to consider and discuss this relevant medical literature, and

[4] It is not unusual for courts to cite to, and consider, published comments underlying the promulgation of regulations. *See Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 156 n. 29 (1988) (favorable discussion of Department's comments underlying promulgation of 20 C.F.R. § 727.203(a) to determine that agency did not intend that a single piece of qualifying evidence would be sufficient to invoke interim presumption); *Consolidation Coal Co. v. Director, OWCP [Stein]*, 294 F.3d 885, 892 (7th Cir. 2002) (favorable consideration of Department's December 2000 comments with regard to use of CT-scans in assessing presence or absence of pneumoconiosis); *Bonessa v. United States Steel Corp.*, 884 F.2d 726, 729 (3rd Cir. 1989) (favorable referral to Department's 1983 comments to 20 C.F.R. § 718.205(c) in assessing causation). Therefore, consideration of the Department's findings as set forth in the comments to the amended regulations is proper here.

to explain why, in this case, he concluded the Miner's COPD was unrelated to his more than 10 years of underground coal mining, diminishes the value of his opinion.

Similarly, while Dr. Tuteur discussed the above-referenced studies, he clearly disagreed with what the Department of Labor and NIOSH considered to be the prevailing view in the medical community concerning the association between coal dust exposure and the development of COPD. EX 11 at 41. For example, Dr. Tuteur testified that various medical studies have led him to conclude that the frequency with which this occurred was "substantially less than one percent." EX 11 at 26-27. He further testified that the frequency with which smokers developed COPD was about 20 percent and he thus concluded that "Mr. Barker's COPD phenotype was due to the chronic inhalation of tobacco smoke . . . ." *Id.* at 27. Dr. Tuteur criticized, *inter alia*, the study of Attfield and Hodous, cited by the Department in its Final Rule, which demonstrated a clear relationship between dust exposure and a decline in pulmonary function of about 5 to 9 milliliters a year, even in miners with no radiographic evidence of clinical coal workers' pneumoconiosis. *Id.* at 59-60. He testified:

> You can take numbers and apply fancy statistics and come up with certain assessments. But when you have a fundamental flaw, which is Experimental Design 101, day one, you need a valid control group. And if you don't have it, no matter what statistics you use, no matter what analysis process you employ, the data cannot be interpreted rigorously. . . .
>
> And I see no evidence in the Federal Register or NIOSH that they have gone through a process such as this, with respect to Attfield and Hodous or other papers, to support their conclusions. They have accepted what Attfield and Hodous says. But I don't – the record is silent with respect to whether they conducted an independent analysis.

Ibid. Dr. Tuteur acknowledged that his disagreements with the methodology and conclusions of Attfield and Hodous had not been published in any peer-reviewed publications for comment. *Id.* at 60.

Given Dr. Tuteur's divergence of opinion with the conclusion reached by the Department of Labor that coal dust exposure may be analogous to the effects of smoking in causing COPD, I find his opinion on whether Mr. Barker's COPD is related to his coal mine employment is entitled to diminished weight. I further note that, in formulating his opinion, Dr. Tuteur relied on a smoking history of the Miner which I find to be greatly exaggerated, a fact which further diminishes the value of his conclusion.[5]

[5] With respect to Dr. Tuteur's findings regarding Mr. Barker's smoking history, I note that he previously assumed a smoking history of one pack of cigarettes per day regarding the Miner's previously denied claim. *See, e.g.*, EX 11 at 46. However, Dr. Tuteur testified in this survivor's case that he now believes the record establishes the Miner smoked up to five packs per day, and that it was quite possible for Mr. Barker to consume five packs of cigarettes per day during the 16 hours he was not working underground as a coal miner. *Id.* at 46, 52. In reaching that conclusion, Dr. Tuteur assumed that Mr. Barker might have "slept a little bit of the time." *Id.* at 52. Evidence in the record substantiates a 20 to 21 year smoking history of one pack of cigarettes per day rather than a five pack-per-day habit. For example, Dr. Robiolio, a treating cardiologist, wrote on October 27, 1995: "As you know, Mr. Barker is a 58-year-old gentleman with cardiac risk factors that include hypertension, tobacco abuse (one pack per day for twenty years, quit in 1979). . . ." DX 14 at 9. Similarly, a pulmonary function study report dated April 15, 1996 notes a 21 year smoking history of 1 pack per day. DX 14 at 77. The Miner's wife also testified that Mr. Barker

In contrast to Drs. Oesterling and Tuteur, Dr. Heidingsfelder, who is board-certified in anatomic pathology, clinical pathology, and forensic pathology, and is a clinical associate professor of pathology at Indiana University Medical Center in Evansville, Indiana, found evidence of, *inter alia*, moderate to marked pulmonary anthracosis and lymph node anthracosis upon macroscopic inspection of the lungs. DX 15 at 7, 73. On microscopic examination of lung tissue, he found evidence of pulmonary emphysema, pulmonary anthracosis with focal interstitial fibrosis, and lymph nodal anthracosis. *Id.* at 13. At his deposition, Dr. Heidingsfelder described his findings when he removed and examined the Miner's lungs:

> The base of the lungs where the lungs attach to the mediastinum revealed anatomic findings of multiple lymph nodes. These appeared to be enlarged, and they were noted on sectioning of the lymph nodes to have a black discoloration of the lymph nodes. The lung tissues in the pleural surface regions and within the underlying lung tissue showed various degrees of moderate to marked anthracotic pigment deposition which was present more or less diffusely in all of the lobes of the lung but was present to a greater degree in the upper lobes on each side. . . .

Id. at 22. He further found "evidence of chronic lung disease including chronic bronchitis and pulmonary emphysema . . . ." *Id.* at 23. With respect to the Miner's emphysema, Dr. Heidingsfelder's microscopic findings revealed "mild to moderate dilation of the air sacs within the lungs near the pleural surface and also underneath the pleural surface within the lung tissue which indicated to me that this person had at lease a moderate degree of pulmonary emphysema. *Id.* at 28. He further testified that microscopic findings "revealed chronic inflammation which is one of the findings you also get in chronic obstructive pulmonary disease referred to as chronic bronchitis." *Ibid*. Tissue slides showed "multiple regions of anthracotic pigment within the lung tissue. According to Dr. Heidingsfelder:

> There was on one section a lesion that was described as a focal anthracotic lesion which is my terminology for a region of localized emphysema around an anthracotic lesion within the parenchyma of the lungs.

Id. at 29. With respect to the causes of these disease processes, Dr. Heidingsfelder testified:

> He has significant degrees of chronic bronchitis; he has significant degrees of pulmonary emphysema, which diseases can be associated with coal worker's pneumoconiosis. There are lesions of localized emphysema which are a more characteristic feature relating to coal workers' pneumoconiosis. It's hard for me to separate out that part which is specifically related to his mining exposure versus other types of exposure that he may have had in his lifetime.

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was smoking about a pack of cigarettes a day in 1973 when they were married and that he quit smoking in June 1979. Tr. 38-40. Other records suggest the possibility of a somewhat greater smoking history. For example, a pulmonary function study report dated December 6, 1994 notes a 22 year smoking history of 2 packs per day. DX 14 at 96. Only one document, a pulmonary function study in January 1999, reflects a 28 year history of smoking five packs per day. DX 13 at 2. Based on the foregoing, I find that the January 1999 pulmonary function report is deserving of little weight and that the Miner smoked, on average, one pack of cigarettes per day for 20 to 21 years.

Id. at 31. He stated that the Miner's emphysema and bronchitis could be attributable to either environmental exposure or smoking. *Id.* at 31-32 He subsequently testified:

> The findings that I made that make me feel that he has coal workers' pneumoconiosis is the presence of other chronic lung diseases of emphysema and bronchitis, the presence of diffuse pleural fibrous scarring, the presence of focal interstitial fibrosis within lung tissues, the presence of marked degrees of the carbon pigment deposits within lung and lymph nodal tissues, *and the presence of lesions of localized emphysema characteristic of simple coal workers' pneumoconiosis*.

Id. at 44-45 (italics added). Dr. Heidingsfelder testified he has not previously seen lesions of localized emphysema characteristic of simple coal workers' pneumoconiosis in individuals who had no history of coal mine dust exposure and had a long history of cigarette smoking exposure. *Id.* at 47. Nor, to his knowledge, does the medical literature suggest that such a finding would occur without exposure to coal dust. *Ibid*. Although he acknowledged that an individual may have carbon deposits within his or her lungs which were unrelated to coal mining exposure, he testified "the particular lesion of localized emphysema [in Mr. Barker's case] is one that's more specific for exposure to coal mining operations." *Id.* at 51. He clarified:

> It's localized emphysema relating to an anthracotic macule. *That is a lesion that is associated with simple coal workers' pneumoconiosis, and the carbon that's related to that lesion particularly in my opinion would originate from the period of [coal mine] exposure*. Now, other carbon that's in the lung – and there was a lot of carbon in the lung, but other carbon in the lung that's not in a specific lesion, I can't say with certainty whether that carbon is derived from environmental exposure or from other exposures.

Id. at 51-52.

Dr. Heidingsfelder, unlike Drs. Oesterling and Tuteur, recognized that both smoking and coal dust exposure can play a substantial role in causing COPD. Dr. Heidingsfelder further noted, consistent with one study cited by the Department which found "strong evidence that emphysema in coalworkers is causally related to lung coal content," Final Rule at 79942, the presence of substantial amounts of carbon throughout the Miner's lungs. Finally, Dr. Heidingsfelder also found specific microscopic evidence associating the Miner's emphysema with coal dust exposure. I find his opinion to be well reasoned and documented in that it is based upon, and consistent with, the macroscopic and microscopic findings set forth in his autopsy report. I thus give it greater weight than the contrary opinions of Drs. Oesterling and Tuteur.

Dr. Green is board-certified in pathology and is a professor of pathology at the University of Calgary, in Calgary, Alberta, Canada. CX 1 at 6. His review of the Miner's autopsy slides revealed "widespread evidence of coal workers' pneumoconiosis, comprising macules, micronodules and interstitial fibrosis with pigmentation." *Id.* at 2. According to Dr. Green:

> The [interstitial fibrosis with pigmentation] consists of numerous particles with a morphology and color of bituminous coal dust as well as birefringent particles consistent with silica and silicates seen on polarizing microscopy. In the vast majority of sections, the number and profusion of the lesions would be consistent with a severe form of simple coal workers' pneumoconiosis. . . . In addition to the pneumoconiosis, there is widespread severe panacinar emphysema, as well as chronic bronchitis involving the large airways and chronic bronchiolitis involving the smaller airways. . . .

Ibid. Based on his review of the Miner's extensive medical records, as well as the autopsy report and deposition testimony of Dr. Heidingsfelder, Dr. Green concluded that Mr. Barker's "pulmonary emphysema was a major component of his chronic lung disease." *Id.* at 3. With respect to the dust found in the Miner's lungs, Dr. Green wrote:

> The dust seen throughout the lung consisted of carbonaceous particles, some of which had the morphologic appearances of combustion products (cigarette smoke, diesel fumes, fire smoke, etc.) and other carbonaceous particles showed the characteristic shape and color of bituminous coal mine dust. In addition, by polarizing microscopy, there were large numbers of particles consistent with silica and silicates. Classic lesions of coal workers' pneumoconiosis were present in Mr. [B]arker's lung, however the pneumoconiosis was distorted by the presence of widespread and destructive emphysema which distorted the lesions of pneumoconiosis to give a more interstitial fibrotic appearance. Interstitial fibrosis is a well-known form of pneumoconiosis in coal miners . . . and gives rise to the more linear (r,s,t) radiologic opacities on the chest x-ray. The simple pneumoconiosis was severe as was the emphysema. Because the emphysema was in an advanced stage, the majority of it is best classified as panacinar; however, panacinar emphysema and bullous emphysema represent the end-stages of all other types of emphysema. . . .

Id. at 3-4. Dr. Green concluded that the Miner's clinical coal workers' pneumoconiosis was caused entirely by exposure to coal mine dust, and, with respect to the cause of his COPD, wrote:

> [It] can be caused by a number of factors, the most common of which are exposure to cigarette smoke and exposure to occupational dusts and fumes. Mr. Barker developed his pulmonary impairment at a relatively young age, and there was a family history of emphysema, raising the possibility that he may have had a genetic predisposition to the development of emphysema. As far as I can tell, the relevant tests were not conducted. In addition to coal mine dust exposure, Mr. Barker also had a history of cigarette smoking, consuming approximately one pack a day for 20 years until he quit in 1979. Numerous epidemiologic and pathologic studies have now unequivocally linked coal mine dust exposure to the development of pulmonary emphysema . . . . Some of these studies have calculated the proportion of emphysema (or reductions in FEV1) attributable to coal dust and cigarette smoking. These show, in general that one pack of smoking contributes approximately the same amount to the emphysema score as does one

> year of underground mining. Using this rule of thumb, it would appear that smoking contributed slightly more to the development of his emphysema than did his exposure to coal mine dust. However, it should be borne in mind that he quit smoking in 1979 and thus for the last 20 years of his life smoking would not further contribute to the development of the emphysema. This would not be true for the relationship with coal mine dust exposure as the dust is retained in the lungs for the lifetime of the individual and continues to irritate the lungs, leading to progression of the emphysema. Taking this into account, I would estimate that smoking and coal mine dust exposure contributed approximately equally to Mr. Barker's emphysema.

Id. at 4.

Dr. Green, like Dr. Heidingsfelder, found the Miner's COPD related to both his smoking and coal dust exposure. Unlike Drs. Oesterling and Tuteur, Dr. Green accepted and relied on the substantial medical literature which reveals a substantial causal relationship between exposure to coal dust and the development of COPD. Dr. Green also based his opinion on what I have found to be an accurate smoking history of the Miner, and recognized that, while the harmful effects of smoking cease to be a factor after the individual stops smoking, the deposition of coal dust in a miner's lungs during his coal mine employment may continue to cause harm well into the future. *See*, *e.g.*, Final Rule at 79968-72 (discussing comments objecting to the Department's rule allowing subsequent claims on the basis that the record lacks adequate justification of the latency and progressivity of pneumoconiosis). I find Dr. Green's opinion to be well reasoned and document and consistent with the similar findings and conclusions of Dr. Heidingsfelder. I thus accord his opinion greater weight than the contrary opinions of Drs. Oesterling and Tuteur.

Based on all the foregoing, I find that a preponderance of the evidence establishes that the Miner had both clinical and legal pneumoconiosis which arose at least in part out of his more than 10 years of employment as an underground coal miner.

C. Causation of Death.

As noted above, an eligible survivor will be entitled to benefits if pneumoconiosis was a substantially contributing cause or factor leading to the miner's death. 20 C.F.R. § 718.205(c). A miner's death will be considered to be due to pneumoconiosis where that disease hastens, even briefly, the miner's death. *Peabody Coal Co. v. Director, OWCP [Riley], supra*.

The Miner's death certificate notes acute myocardial infarction as the immediate cause of death and lists pneumoconiosis as a significant condition contributing to death. DX 9. Dr. Heidingsfelder concurred in this assessment, testifying that, based on his examination of Mr. Barker's heart and lungs, it was his opinion that the Miner died of a heart attack which occurred over a period of days or weeks and that chronic lung disease, which included emphysema, bronchitis, and pneumoconiosis all working together, was a contributing factor to the timing of his heart attack. DX 15 at 67-68. Dr. Tuteur similarly recognized that, "[c]omplicating [the Miner's] death almost certainly was the advanced chronic pulmonary emphysema despite functional improvement after lung volume reduction surgery." EX 9 at 5. Dr. Green similarly

agreed with the death certificate and Dr. Heidingsfelder, stating that "chronic lung disease was a contributing factor to the [Miner's] death." CX 1 at 3. Although Dr. Oesterling concluded that pneumoconiosis was not a substantial contributing cause in the Miner's death, EX 1 at 2, this conclusion was based on his finding of clinical, but not legal, pneumoconiosis, a conclusion which, as noted above, I have found to be contrary to the better reasoned opinions of Drs. Green and Heidingsfelder. Furthermore, Dr. Green has clearly acknowledged that Mr. Barker "appeared to be [in] significant respiratory distress [in his more terminal period prior to death]." *Id.* at 5. According to the Board:

> We note that as the Secretary observed when promulgating Section 718.205(c)(5), the proposition that persons weakened by pneumoconiosis may expire quicker from other diseases *is* a medical point, with some empirical support. *See* 65 Fed. Reg. 79,920, 79,950 (Dec. 20, 2000).

Bailey v. Consolidation Coal Co., BRB No. 05-0324 BLA (Sept. 30, 2005) (unpub.), slip op. at 6 (emphasis in original).

Based on all the foregoing, I find that the weight of the medical evidence supports the conclusion that Mr. Barker's pneumoconiosis was a substantially contributing cause of his death. I thus find that Claimant is entitled to an award of benefits as the Miner's eligible survivor.

Attorney's Fees

No award of attorney's fees for services to Claimant is made herein, as no application has yet been received from her representative. Thirty days are hereby allowed to Claimant's counsel for the submission of such application. Her attention is directed to §§ 725.365 and 725.366 of the Regulations. A service sheet showing service upon all parties, including the Claimant, must accompany the application. Parties have fifteen days following receipt of such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claim of KATHRYN S. BARKER, survivor of JAMES A. BARKER, for benefits under the Act, is hereby GRANTED, and it is hereby ORDERED that PEABODY COAL COMPANY, the Responsible Operator, shall pay to Claimant all benefits to which she is entitled under the Act, commencing June 7, 2003.

A

STEPHEN L. PURCELL
Administrative Law Judge

Washington, D.C.

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Order may appeal it to the Benefits Review within 30 days from the date of this Order by filing a Notice of Appeal with the Benefits Review Board, P.O. Box 37601, Washington DC 20013- 7601. A copy of a Notice of Appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits. His address is Room N-2117, Frances Perkins Building, 200 Constitution Avenue, N.W., Washington DC 20210.